General

Guideline Title

2013 ACCF/AHA guideline for the management of heart failure. A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines.

Bibliographic Source(s)

Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Drazner MH, Fonarow GC, Geraci SA, Horwich T, Januzzi JL, Johnson MR, Kasper EK, Levy WC, Masoudi FA, McBride PE, McMurray JJ, Mitchell JE, Peterson PN, Riegel B, Sam F, Stevenson LW, Tang WH, Tsai EJ, Wilkoff BL. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2013 Oct 15;62(16):e147-239. [924 references] PubMed

Guideline Status

This is the current release of the guideline.

This guideline updates a previous version: Hunt SA, Abraham WT, Chin MH, Feldman AM, Francis GS, Ganiats TG, Jessup M, Konstam MA, Mancini DM, Michl K, Oates JA, Rahko PS, Silver MA, Stevenson LW, Yancy CW, American College of Cardiology Foundation, American Heart Association. 2009 focused update incorporated into the ACC/AHA 2005 guidelines for the diagnosis and management of heart failure in adults [trunc]. J Am Coll Cardiol. 2009 Apr 14;53(15):e1-e90.

Recommendations

Major Recommendations

Definitions for the levels of the evidence (A-C) and classes of recommendations (I-III) are provided at the end of the "Major Recommendations" field.

Initial and Serial Evaluation of the Heart Failure (HF) Patient

Clinical Evaluation

History and Physical Examinations

Class I

1. A thorough history and physical examination should be obtained/performed in patients presenting with HF to identify cardiac and noncardiac disorders or behaviors that might cause or accelerate the development or progression of HF. (*Level of Evidence: C*)

2. In patients with idiopathic dilated cardiomyopathy (DCM), a 3-generational family history should be obtained to aid in establishing the diagnosis of familial DCM. (*Level of Evidence: C*)
3. Volume status and vital signs should be assessed at each patient encounter. This includes serial assessment of weight, as well as estimates of jugular venous pressure and the presence of peripheral edema or orthopnea (Butman et al., 1993; Drazner et al., 2001; Drazner et al., 2008; Stevenson & Perloff, 1989). (*Level of Evidence: B*)

Risk Scoring

Class IIa

1. Validated multivariable risk scores can be useful to estimate subsequent risk of mortality in ambulatory or hospitalized patients with HF (Aaronson et al., 1997; Fonarow et al., 2005; Komajda et al., 2011; Lee et al., 2003; Levy et al., 2006; O'Connor et al., 2008; Peterson et al., 2010; Pocock et al., 2006; Wedel et al., 2009). (*Level of Evidence: B*)

Diagnostic Tests

Class I

1. Initial laboratory evaluation of patients presenting with HF should include complete blood count, urinalysis, serum electrolytes (including calcium and magnesium), blood urea nitrogen, serum creatinine, glucose, fasting lipid profile, liver function tests, and thyroid-stimulating hormone. (*Level of Evidence: C*)
2. Serial monitoring, when indicated, should include serum electrolytes and renal function. (*Level of Evidence: C*)
3. A 12-lead electrocardiogram (ECG) should be performed initially on all patients presenting with HF. (*Level of Evidence: C*)

Class IIa

1. Screening for hemochromatosis or human immunodeficiency virus (HIV) is reasonable in selected patients who present with HF (Okonko et al., 2011). (*Level of Evidence: C*)
2. Diagnostic tests for rheumatologic diseases, amyloidosis, or pheochromocytoma are reasonable in patients presenting with HF in whom there is a clinical suspicion of these diseases. (*Level of Evidence: C*)

Biomarkers

Ambulatory/Outpatient

Class I

1. In ambulatory patients with dyspnea, measurement of B-type natriuretic peptide (BNP) or N-terminal pro-B-type natriuretic peptide (NT-proBNP) is useful to support clinical decision making regarding the diagnosis of HF, especially in the setting of clinical uncertainty (Costello-Boerrigter et al., 2006; de Lemos et al., 2009; Goetze et al., 2006; Ng et al., 2005; Richards et al., 2001; Tang et al., 2003; Vasan et al., 2002). (*Level of Evidence: A*)
2. Measurement of BNP or NT-proBNP is useful for establishing prognosis or disease severity in chronic HF (Tang et al., 2003; Berger et al., 2002; Anand et al., 2003; Forfia et al., 2005; Taub, Daniels, & Maisel, 2009; Maeda et al., 2000; Neuhold et al., 2008). (*Level of Evidence: A*)

Class IIa

1. BNP- or NT-proBNP–guided HF therapy can be useful to achieve optimal dosing of guideline-directed medical therapy (GDMT) in select clinically euvolemic patients followed in a well-structured HF disease management program (Januzzi et al., 2011; Porapakkham et al., 2010; Felker et al., 2009; Jourdain et al., 2007; Pfisterer et al., 2009; Berger et al., 2010; Troughton et al., 2000; Lainchbury et al., 2009). (*Level of Evidence: B*)

Class IIb

1. The usefulness of serial measurement of BNP or NT-proBNP to reduce hospitalization or mortality in patients with HF is not well established (Januzzi et al., 2011; Porapakkham et al., 2010; Felker et al., 2009; Jourdain et al., 2007; Pfisterer et al., 2009; Berger et al., 2010; Troughton et al., 2000; Lainchbury et al., 2009). (*Level of Evidence: B*)
2. Measurement of other clinically available tests such as biomarkers of myocardial injury or fibrosis may be considered for additive risk stratification in patients with chronic HF (Horwich et al., 2003; Sato et al., 2001; Setsuta et al., 1999; Hudson et al., 2004; Tang et al., 2011; de Boer et al., 2011; Lok et al., 2010). (*Level of Evidence: B*)

Hospitalized/Acute

Class I

1. Measurement of BNP or NT-proBNP is useful to support clinical judgment for the diagnosis of acutely decompensated HF, especially in the setting of uncertainty for the diagnosis (Januzzi et al., 2006; Dao et al., 2001; Davis et al., 1994; Maisel et al., 2002; van Kimmenade et al., 2006; Moe et al., 2007; Mueller et al., 2004). (*Level of Evidence: A*)
2. Measurement of BNP or NT-proBNP and/or cardiac troponin is useful for establishing prognosis or disease severity in acutely decompensated HF (van Kimmenade et al., 2006; Bettencourt et al., 2004; Cheng et al., 2001; Fonarow et al. "Usefulness," 2008; Logeart et al., 2004; Maisel et al., 2004; Zairis et al., 2010; Peacock et al., 2008; Lee et al., 2012). (*Level of Evidence: A*)

Class IIb

1. The usefulness of BNP- or NT-proBNP–guided therapy for acutely decompensated HF is not well established (Bayes-Genis et al., 2005; Dhaliwal et al., 2009). (*Level of Evidence: C*)
2. Measurement of other clinically available tests such as biomarkers of myocardial injury or fibrosis may be considered for additive risk stratification in patients with acutely decompensated HF (van Kimmenade et al., 2006; Fonarow et al. "Influence," 2008; Zairis et al., 2010; Peacock et al., 2008; Alonso-Martinez et al., 2002; Dieplinger et al., 2010; Ilva et al., 2008; Januzzi et al., 2007; Manzano-Fernandez et al., 2011; Rehman et al., 2008; Shah et al., 2010). (*Level of Evidence:* A)

Noninvasive Cardiac Imaging

Class I

1. Patients with suspected or new-onset HF, or those presenting with acute decompensated HF, should undergo a chest x-ray to assess heart size and pulmonary congestion and to detect alternative cardiac, pulmonary, and other diseases that may cause or contribute to the patient's symptoms. (*Level of Evidence: C*)
2. A 2-dimensional echocardiogram with Doppler should be performed during initial evaluation of patients presenting with HF to assess ventricular function, size, wall thickness, wall motion, and valve function. (*Level of Evidence: C*)
3. Repeat measurement of ejection fraction (EF) and measurement of the severity of structural remodeling are useful to provide information in patients with HF who have had a significant change in clinical status; who have experienced or recovered from a clinical event; or who have received treatment, including GDMT, that might have had a significant effect on cardiac function; or who may be candidates for device therapy. (*Level of Evidence: C*)

Class IIa

1. Noninvasive imaging to detect myocardial ischemia and viability is reasonable in patients presenting with de novo HF, who have known coronary artery disease (CAD) and no angina, unless the patient is not eligible for revascularization of any kind. (*Level of Evidence: C*)
2. Viability assessment is reasonable in select situations when planning revascularization in HF patients with CAD (Rizzello et al., 2009; Allman et al., 2002; Beanlands et al., 2002; Pagley et al. 1997; Senior, Kaul, & Lahiri, 1999). (*Level of Evidence: B*)
3. Radionuclide ventriculography or magnetic resonance imaging can be useful to assess left ventricular ejection fraction (LVEF) and volume when echocardiography is inadequate. (*Level of Evidence: C*)
4. Magnetic resonance imaging is reasonable when assessing myocardial infiltrative processes or scar burden (Kwon et al., 2009; Ordovas & Higgins, 2011; Syed et al., 2010). (*Level of Evidence:* B)

Class III: No Benefit

1. Routine repeat measurement of LV function assessment in the absence of clinical status change or treatment interventions should not be performed (Bellar, 2012; American College of Cardiology Foundation Appropriate Use Criteria Task Force et al., 2011). (*Level of Evidence: B*)

Invasive Evaluation

Class I

1. Invasive hemodynamic monitoring with a pulmonary artery catheter should be performed to guide therapy in patients who have respiratory distress or clinical evidence of impaired perfusion in whom the adequacy or excess of intracardiac filling pressures cannot be determined from clinical assessment. (*Level of Evidence: C*)

Class IIa

1. Invasive hemodynamic monitoring can be useful for carefully selected patients with acute HF who have persistent symptoms despite empiric adjustment of standard therapies and
    a. Whose fluid status, perfusion, or systemic or pulmonary vascular resistance is uncertain
    b. Whose systolic pressure remains low, or is associated with symptoms, despite initial therapy
    c. Whose renal function is worsening with therapy
    d. Who require parenteral vasoactive agents
    e. Who may need consideration for mechanical circulatory support (MCS) or transplantation (*Level of Evidence: C*)

2. When ischemia may be contributing to HF, coronary arteriography is reasonable for patients eligible for revascularization. (*Level of Evidence: C*)
3. Endomyocardial biopsy can be useful in patients presenting with HF when a specific diagnosis is suspected that would influence therapy. (*Level of Evidence: C*)

Class III: No Benefit

1. Routine use of invasive hemodynamic monitoring is not recommended in normotensive patients with acute decompensated HF and congestion with symptomatic response to diuretics and vasodilators (Binanay et al., 2005). (*Level of Evidence: B*)

Class III: Harm

1. Endomyocardial biopsy should not be performed in the routine evaluation of patients with HF. (*Level of Evidence: C*)

Treatment of Stages A to D

Stage A

Class I

1. Hypertension and lipid disorders should be controlled in accordance with contemporary guidelines to lower the risk of HF (Chobanian et al., 2003; Kostis et al., 1997; Beckett et al., 2008; Sciarretta et al., 2011; Staessen, Wang, & Thijs, 2003; Verdecchia et al., 2009). (*Level of Evidence: A*)
2. Other conditions that may lead to or contribute to HF, such as obesity, diabetes mellitus, tobacco use, and known cardiotoxic agents, should be controlled or avoided. (*Level of Evidence: C*)

Stage B

Class I

1. In all patients with a recent or remote history of myocardial infarction (MI) or acute coronary syndrome (ACS) and reduced EF, angiotensin-converting-enzyme (ACE) inhibitors should be used to prevent symptomatic HF and reduce mortality (Pfeffer et al., 1992; CONSENSUS Trial Study Group, 1987; "Effect of enalapril on mortality," 1992). In patients intolerant of ACE inhibitors, angiotensin receptor blockers (ARBs) are appropriate unless contraindicated (Verdecchia et al., 2009; Pfeffer et al., "Valsartan," 2003). (*Level of Evidence: A*)
2. In all patients with a recent or remote history of MI or ACS and reduced EF, evidence-based beta blockers should be used to reduce mortality (Dargie, 2001; Vantrimpont et al., 1997; Exner et al., 1999). (*Level of Evidence: B*)
3. In all patients with a recent or remote history of MI or ACS, statins should be used to prevent symptomatic HF and cardiovascular events (Grundy et al., 2004; Scirica et al., 2006; Afilalo, Majdan, & Eisenberg, 2007; Ho et al., 2012; Strandberg et al., 2009; Kjekshus et al., 1997; Sacks et al., 1996). (*Level of Evidence: A*)
4. In patients with structural cardiac abnormalities, including LV hypertrophy, in the absence of a history of MI or ACS, blood pressure should be controlled in accordance with clinical practice guidelines for hypertension to prevent symptomatic HF (Chobanian et al., 2003; Kostis et al., 1997; Beckett et al., 2008; Sciarretta et al., 2011; Staessen, Wang, & Thijs, 2003). (*Level of Evidence: A*)
5. ACE inhibitors should be used in all patients with a reduced EF to prevent symptomatic HF, even if they do not have a history of MI (Jong et al., 2003; "Effect of enalapril on mortality," 1992). (*Level of Evidence: A*)
6. Beta blockers should be used in all patients with a reduced EF to prevent symptomatic HF, even if they do not have a history of MI. (*Level of Evidence: C*)

Class IIa

1. To prevent sudden death, placement of an implantable cardioverter-defibrillator (ICD) is reasonable in patients with asymptomatic ischemic cardiomyopathy who are at least 40 days post-MI, have an LVEF of 30% or less, are on appropriate medical therapy, and have reasonable expectation of survival with a good functional status for more than 1 year (Moss et al., 2002). (*Level of Evidence: B*)

Class III: Harm

1. Nondihydropyridine calcium channel blockers with negative inotropic effects may be harmful in asymptomatic patients with low LVEF and no symptoms of HF after MI. (*Level of Evidence: C*)

Stage C

Nonpharmacological Interventions—Education

Class I

1. Patients with HF should receive specific education to facilitate HF self-care (Boren et al., 2009; Gwadry-Sridhar et al., 2005; Koelling et al., 2005; VanSuch et al., 2006; Aguado et al., 2010; Riegel et al., 2009). (*Level of Evidence: B*)

Nonpharmacological Interventions—Sodium Restriction

Class IIa

1. Sodium restriction is reasonable for patients with symptomatic HF to reduce congestive symptoms. (*Level of Evidence: C*)

Nonpharmacological Interventions—Treatment of Sleep Disorders

Class IIa

1. Continuous positive airway pressure can be beneficial to increase LVEF and improve functional status in patients with HF and sleep apnea (Arzt et al., 2007; Bradley et al., 2005; Kaneko et al., 2003; Mansfield et al., 2004). (*Level of Evidence: B*)

Nonpharmacological Interventions—Activity, Exercise Prescription, and Cardiac Rehabilitation

Class I

1. Exercise training (or regular physical activity) is recommended as safe and effective for patients with HF who are able to participate to improve functional status (Davies et al., 2010; McKelvie, 2008; O'Connor et al., 2009; Pina et al., 2003). (*Level of Evidence: A*)

Class IIa

1. Cardiac rehabilitation can be useful in clinically stable patients with HF to improve functional capacity, exercise duration, health-related quality of life (HRQOL), and mortality (Davies et al., 2010; O'Connor et al., 2009; Pina et al., 2003; Smart & Marwick, 2004; Piepoli et al., 2004; Austin et al., 2005; Austin et al., 2008). (*Level of Evidence: B*)

Pharmacological Treatment for Stage C Heart Failure with Reduced Ejection Fraction (HFrEF)

Class I

1. Measures listed as Class I recommendations for patients in stages A and B are recommended where appropriate for patients in stage C. (*Levels of Evidence: A, B, and C as appropriate*)
2. GDMT as depicted in Figure 1 in the original guideline document should be the mainstay of pharmacological therapy for HF*r*EF (Cohn & Tognoni, 2001; CONSENSUS Trial Study Group, 1987; Pfeffer et al., "Valsartan," 2003; Dargie, 2001; Cohn et al., 1991; SOLVD Investigators, 1991; Garg & Yusuf, 1995; Maggioni et al., 2002; "Xamoterol in severe heart failure," 1990; "Effects of carvedilol," 1995; Beta-Blocker Evaluation of Survival Trial Investigators, 2001; Poole-Wilson et al., 2003; Pfeffer et al., "Effects," 2003; Konstam et al., 2009; Pitt et al., 1997; Carson et al., 1999; Taylor et al., 2004; Pitt et al., 1999; Zannad et al., 2011). (*Level of Evidence: A*)

Pharmacological Treatment for Stage C HFrEF—Diuretics

Class I

1. Diuretics are recommended in patients with HF*r*EF who have evidence of fluid retention, unless contraindicated, to improve symptoms. (*Level of Evidence: C*)

Pharmacological Treatment for Stage C HFrEF—ACE Inhibitors

Class I

1. ACE inhibitors are recommended in patients with HF*r*EF and current or prior symptoms, unless contraindicated, to reduce morbidity and mortality (CONSENSUS Trial Study Group, 1987; Cohn et al., 1991; SOLVD Investigators, 1991; Garg & Yusuf, 1995). (*Level of Evidence: A*)

Pharmacological Treatment for Stage C HFrEF—ARBs

Class I

1. ARBs are recommended in patients with HF*r*EF with current or prior symptoms who are ACE inhibitor intolerant, unless contraindicated, to reduce morbidity and mortality (Cohn & Tognoni, 2001; Pfeffer et al., "Valsartan," 2003; Maggioni et al., 2002; Granger et al., 2003). (*Level of Evidence: A*)

Class IIa

1. ARBs are reasonable to reduce morbidity and mortality as alternatives to ACE inhibitors as first-line therapy for patients with HF*r*EF, especially for patients already taking ARBs for other indications, unless contraindicated (Crozier et al., 1995; Gottlieb et al., 1993; Mazayev et al., 1998; McKelvie et al., 1999; Riegger et al., 1999; Sharma et al., 2000). (*Level of Evidence: A*)

Class IIb

1. Addition of an ARB may be considered in persistently symptomatic patients with HF*r*EF who are already being treated with an ACE inhibitor and a beta blocker in whom an aldosterone antagonist is not indicated or tolerated (Pfeffer et al., "Effects," 2003; Velazquez et al., 2003). (*Level of Evidence: A*)

Class III: Harm

1. Routine combined use of an ACE inhibitor, ARB, and aldosterone antagonist is potentially harmful for patients with HF*r*EF. (*Level of Evidence: C*)

Pharmacological Treatment for Stage C HFrEF—Beta Blockers

Class I

1. Use of 1 of the 3 beta blockers proven to reduce mortality (e.g., bisoprolol, carvedilol, and sustained-release metoprolol succinate) is recommended for all patients with current or prior symptoms of HF*r*EF, unless contraindicated, to reduce morbidity and mortality (Dargie, 2001; "Xamoterol in severe heart failure," 1990; "Effects of carvedilol," 1995; Beta-Blocker Evaluation of Survival Trial Investigators, 2001; Poole-Wilson et al., 2003; "Effect of metoprolol CR/XL in chronic heart failure," 1999). (*Level of Evidence: A*)

Pharmacological Treatment for Stage C HFrEF—Aldosterone Receptor Antagonists

Class I

1. Aldosterone receptor antagonists (or mineralocorticoid receptor antagonists) are recommended in patients with New York Heart Association (NYHA) class II–IV HF and who have LVEF of 35% or less, unless contraindicated, to reduce morbidity and mortality. Patients with NYHA class II HF should have a history of prior cardiovascular hospitalization or elevated plasma natriuretic peptide levels to be considered for aldosterone receptor antagonists. Creatinine should be 2.5 mg/dL or less in men or 2.0 mg/dL or less in women (or estimated glomerular filtration rate >30 mL/min/1.73 m^2), and potassium should be less than 5.0 mEq/L. Careful monitoring of potassium, renal function, and diuretic dosing should be performed at initiation and closely followed thereafter to minimize risk of hyperkalemia and renal insufficiency (Pitt et al., 1999; Zannad et al., 2011; Vizzardi et al., 2010). (*Level of Evidence: A*)
2. Aldosterone receptor antagonists are recommended to reduce morbidity and mortality following an acute MI in patients who have LVEF of 40% or less who develop symptoms of HF or who have a history of diabetes mellitus, unless contraindicated (Pitt et al., 2003). (*Level of Evidence: B*)

Class III: Harm

1. Inappropriate use of aldosterone receptor antagonists is potentially harmful because of life-threatening hyperkalemia or renal insufficiency

when serum creatinine is greater than 2.5 mg/dL in men or greater than 2.0 mg/dL in women (or estimated glomerular filtration rate <30 mL/min/1.73 m^2), and/or potassium greater than 5.0 mEq/L (Juurlink et al., 2004; Bozkurt, Agoston, & Knowlton, 2003). (*Level of Evidence: B*)

Pharmacological Treatment for Stage C HFrEF—Hydralazine and Isosorbide Dinitrates

Class I

1. The combination of hydralazine and isosorbide dinitrate is recommended to reduce morbidity and mortality for patients self-described as African Americans with NYHA class III–IV HF*r*EF receiving optimal therapy with ACE inhibitors and beta blockers, unless contraindicated (Carson et al., 1999; Taylor et al., 2004). (*Level of Evidence: A*)

Class IIa

1. A combination of hydralazine and isosorbide dinitrate can be useful to reduce morbidity or mortality in patients with current or prior symptomatic HF*r*EF who cannot be given an ACE inhibitor or ARB because of drug intolerance, hypotension, or renal insufficiency, unless contraindicated (Cohn et al., 1986). (*Level of Evidence: B*)

Pharmacological Treatment for Stage C HFrEF—Digoxin

Class IIa

1. Digoxin can be beneficial in patients with HF*r*EF, unless contraindicated, to decrease hospitalizations for HF (Digitalis Investigation Group, 1997; "Comparative effects of therapy," 1988; Dobbs, Kenyon, & Dobbs, 1977; Lee et al., 1982; Guyatt et al., 1988; DiBianco et al., 1989; Uretsky et al., 1993; Packer et al., 1993). (*Level of Evidence: B*)

Pharmacological Treatment for Stage C HFrEF—Other Drug Treatment: Anticoagulation

Class I

1. Patients with chronic HF with permanent/persistent/paroxysmal atrial fibrillation (AF) and an additional risk factor for cardioembolic stroke (history of hypertension, diabetes mellitus, previous stroke or transient ischemic attack, or ≥75 years of age) should receive chronic anticoagulant therapy* (Granger et al., 2011; Cairns et al., 2011; "Risk factors for stroke and efficacy of antithrombotic therapy in atrial fibrillation," 1994; Hughes & Lip, 2008; Connolly et al., 2009; Connolly et al., 2010; Patel et al., 2011). (*Level of Evidence: A*)
2. The selection of an anticoagulant agent (warfarin, dabigatran, apixaban, or rivaroxaban) for permanent/persistent/paroxysmal AF should be individualized on the basis of risk factors, cost, tolerability, patient preference, potential for drug interactions, and other clinical characteristics, including time in the international normalized ratio therapeutic range if the patient has been taking warfarin. (*Level of Evidence: C*)

Class IIa

1. Chronic anticoagulation is reasonable for patients with chronic HF who have permanent/persistent/paroxysmal AF but are without an additional risk factor for cardioembolic stroke* (Cairns et al., 2011; "Risk factors for stroke and efficacy of antithrombotic therapy in atrial fibrillation," 1994; Hughes & Lip, 2008; Dries et al., 1997; European Heart Rhythm Association et al., 2010; Freudenberger et al., 2007). (*Level of Evidence: B*)

*In the absence of contraindications to anticoagulation.

Class III: No Benefit

1. Anticoagulation is not recommended in patients with chronic HF*r*EF without AF, a prior thromboembolic event, or a cardioembolic source (Loh et al., 1997; Massie et al., 2009; Homma et al., 2012). (*Level of Evidence: B*)

Pharmacological Treatment for Stage C HFrEF—Other Drug Treatment: Statins

Class III: No Benefit

1. Statins are not beneficial as adjunctive therapy when prescribed solely for the diagnosis of HF in the absence of other indications for their use (Horwich, MacLellan, & Fonarow, 2004; Anker et al., 2006; Go et al., 2006; Foody et al., 2006; Kjekshus et al., 2007; GISSI-HF Investigators et al., "Effect of rosuvastatin," 2008). (*Level of Evidence: A*)

Pharmacological Treatment for Stage C HFrEF—Other Drug Treatment: Omega-3 Fatty Acids

Class IIa

1. Omega-3 polyunsaturated fatty acid (PUFA) supplementation is reasonable to use as adjunctive therapy in patients with NYHA class II–IV symptoms and HF*r*EF or heart failure with preserved ejection fraction (HF*p*EF), unless contraindicated, to reduce mortality and cardiovascular hospitalizations (Macchia et al., 2005; GISSI-HF Investigators et al., "Effect of n-3," 2008). (*Level of Evidence: B*)

Pharmacological Treatment for Stage C HFrEF—Other Drug Treatment: Drugs of Unproven Value or That May Worsen HF

Class III: No Benefit

1. Nutritional supplements as treatment for HF are not recommended in patients with current or prior symptoms of HF*r*EF (McMurray et al., 2010; Soukoulis et al., 2009). (*Level of Evidence: B*)
2. Hormonal therapies other than to correct deficiencies are not recommended for patients with current or prior symptoms of HF*r*EF. (*Level of Evidence: C*)

Class III: Harm

1. Drugs known to adversely affect the clinical status of patients with current or prior symptoms of HF*r*EF are potentially harmful and should be avoided or withdrawn whenever possible (e.g., most antiarrhythmic drugs, most calcium channel–blocking drugs [except amlodipine], nonsteroidal anti-inflammatory drugs [NSAIDs], or thiazolidinediones) ("Effect of verapamil on mortality and major events," 1990; Goldstein et al., 1991; Waldo et al., 1996; Kober et al., 2008; "Preliminary report," 1989; "The effect of diltiazem," 1988; Figulla et al., 1996; Elkayam et al., 1990; Gislason et al., 2009; Heerdink et al., 1998; Hudson, Richard, & Pilote, 2005; Lipscombe et al., 2007). (*Level of Evidence: B*)
2. Long-term use of infused positive inotropic drugs is potentially harmful for patients with HF*r*EF, except as palliation for patients with end-stage disease who cannot be stabilized with standard medical treatment (see recommendations for stage D). (*Level of Evidence: C*)

Pharmacological Treatment for Stage C HFrEF—Other Drug Treatment: Calcium Channel Blockers

Class III: No Benefit

1. Calcium channel–blocking drugs are not recommended as routine treatment for patients with HF*r*EF ("The effect of diltiazem," 1988; Setaro et al., 1990; Packer et al., 1996). (*Level of Evidence: A*)

Pharmacological Treatment for Stage C HFpEF

Class I

1. Systolic and diastolic blood pressure should be controlled in patients with HF*p*EF in accordance with published clinical practice guidelines to prevent morbidity (Chobanian et al., 2003; Levy et al., 1996). (*Level of Evidence: B*)
2. Diuretics should be used for relief of symptoms due to volume overload in patients with HF*p*EF. (*Level of Evidence: C*)

Class IIa

1. Coronary revascularization is reasonable in patients with CAD in whom symptoms (angina) or demonstrable myocardial ischemia is judged to be having an adverse effect on symptomatic HF*p*EF despite GDMT. (*Level of Evidence: C*)
2. Management of AF according to published clinical practice guidelines in patients with HF*p*EF is reasonable to improve symptomatic HF (see Section 9.1 in the original guideline document). (*Level of Evidence: C*)
3. The use of beta-blocking agents, ACE inhibitors, and ARBs in patients with hypertension is reasonable to control blood pressure in patients with HF*p*EF. (*Level of Evidence: C*)

Class IIb

1. The use of ARBs might be considered to decrease hospitalizations for patients with HF*p*EF (Yusuf et al., 2003). (*Level of Evidence: B*)

Class III: No Benefit

1. Routine use of nutritional supplements is not recommended for patients with HF*p*EF. (*Level of Evidence: C*)

Device Therapy for Stage C HFrEF

Class I

1. ICD therapy is recommended for primary prevention of sudden cardiac death (SCD) to reduce total mortality in selected patients with nonischemic DCM or ischemic heart disease at least 40 days post-MI with LVEF of 35% or less and NYHA class II or III symptoms on chronic GDMT, who have reasonable expectation of meaningful survival for more than 1 year† (Moss et al., 2002; Bardy et al., 2005). (*Level of Evidence: A*)
2. Cardiac resynchronization therapy (CRT) is indicated for patients who have LVEF of 35% or less, sinus rhythm, left bundle-branch block (LBBB) with a QRS duration of 150 ms or greater, and NYHA class II, III, or ambulatory IV symptoms on GDMT. (*Level of Evidence: A for NYHA class III/IV* [Hunt et al., 2009; Cleland et al., 2005; Bristow et al., 2004; Abraham et al., 2002]; *Level of Evidence: B for NYHA class II* [Moss et al., 2009; Tang et al., 2010]).
3. ICD therapy is recommended for primary prevention of SCD to reduce total mortality in selected patients at least 40 days post-MI with LVEF of 30% or less, and NYHA class I symptoms while receiving GDMT, who have reasonable expectation of meaningful survival for more than 1 year† (Moss et al., 1996; Buxton et al., 1999; Hohnloser et al., 2004). (*Level of Evidence: B*)

†Counseling should be specific to each individual patient and should include documentation of a discussion about the potential for sudden death and nonsudden death from HF or noncardiac conditions. Information should be provided about the efficacy, safety, and potential complications of an ICD and the potential for defibrillation to be inactivated if desired in the future, notably when a patient is approaching end of life. This will facilitate shared decision making between patients, families, and the medical care team about ICDs (Allen et al., 2012).

Class IIa

1. CRT can be useful for patients who have LVEF of 35% or less, sinus rhythm, a non-left bundle branch block (LBBB) pattern with a QRS duration of 150 ms or greater, and NYHA class III/ambulatory class IV symptoms on GDMT (Cleland et al., 2005; Bristow et al., 2004; Abraham et al., 2002; Tang et al., 2010). (*Level of Evidence: A*)
2. CRT can be useful for patients who have LVEF of 35% or less, sinus rhythm, LBBB with a QRS duration of 120 to 149 ms, and NYHA class II, III, or ambulatory IV symptoms on GDMT (Cleland et al., 2005; Bristow et al., 2004; Abraham et al., 2002; Moss et al., 2009; Tang et al., 2010; Linde et al., 2008). (*Level of Evidence: B*)
3. CRT can be useful in patients with AF and LVEF of 35% or less on GDMT if a) the patient requires ventricular pacing or otherwise meets CRT criteria and b) atrioventricular nodal ablation or pharmacological rate control will allow near 100% ventricular pacing with CRT (Brignole et al., 2005; Brignole et al., 2011; Doshi et al., 2005; Gasparini et al., 2006; Wilton et al., 2011; Upadhyay et al., 2008). (*Level of Evidence: B*)
4. CRT can be useful for patients on GDMT who have LVEF of 35% or less and are undergoing placement of a new or replacement device implantation with anticipated requirement for significant (>40%) ventricular pacing (Wilkoff et al., 2002; Doshi et al., 2005; Adelstein et al., 2011; Vatankulu et al., 2009) . (*Level of Evidence: C*)

Class IIb

1. The usefulness of implantation of an ICD is of uncertain benefit to prolong meaningful survival in patients with a high risk of nonsudden death as predicted by frequent hospitalizations, advanced frailty, or comorbidities such as systemic malignancy or severe renal dysfunction† (Setoguchi et al., 2009; Carson et al., 2005; Zareba et al., 2005; Mozaffarian et al., 2007). (*Level of Evidence: B*)
2. CRT may be considered for patients who have LVEF of 35% or less, sinus rhythm, a non-LBBB pattern with QRS duration of 120 to 149 ms, and NYHA class III/ambulatory class IV on GDMT (Tang et al., 2010; Rickard et al., 2011). (*Level of Evidence: B*)
3. CRT may be considered for patients who have LVEF of 35% or less, sinus rhythm, a non-LBBB pattern with a QRS duration of 150 ms or greater, and NYHA class II symptoms on GDMT (Moss et al., 2009; Tang et al., 2010). (*Level of Evidence: B*)
4. CRT may be considered for patients who have LVEF of 30% or less, ischemic etiology of HF, sinus rhythm, LBBB with a QRS duration of 150 ms or greater, and NYHA class I symptoms on GDMT (Moss et al., 2009; Tang et al., 2010). (*Level of Evidence: C*)

†Counseling should be specific to each individual patient and should include documentation of a discussion about the potential for sudden death and nonsudden death from HF or noncardiac conditions. Information should be provided about the efficacy, safety, and potential complications of an ICD and the potential for defibrillation to be inactivated if desired in the future, notably when a patient is approaching end of life. This will facilitate shared decision making between patients, families, and the medical care team about ICDs (Allen et al., 2012).

Class III: No Benefit

1. CRT is not recommended for patients with NYHA class I or II symptoms and non-LBBB pattern with QRS duration less than 150 ms (Moss et al., 2009; Tang et al., 2010; Rickard et al., 2011). (*Level of Evidence: B*)
2. CRT is not indicated for patients whose comorbidities and/or frailty limit survival with good functional capacity to less than 1 year (Hunt et

al., 2009). (*Level of Evidence: C*)

See Figure 2, "Indications for CRT therapy algorithm," in the original guideline document.

Stage D

Water Restriction

Class IIa

1. Fluid restriction (1.5 to 2 L/d) is reasonable in stage D, especially in patients with hyponatremia, to reduce congestive symptoms. (*Level of Evidence: C*)

Inotropic Support

Class I

1. Until definitive therapy (e.g., coronary revascularization, MCS, heart transplantation) or resolution of the acute precipitating problem, patients with cardiogenic shock should receive temporary intravenous inotropic support to maintain systemic perfusion and preserve end-organ performance. (*Level of Evidence: C*)

Class IIa

1. Continuous intravenous inotropic support is reasonable as "bridge therapy" in patients with stage D HF refractory to GDMT and device therapy who are eligible for and awaiting MCS or cardiac transplantation (Aranda et al., 2003; Brozena et al., 2004). (*Level of Evidence: B*)

Class IIb

1. Short-term, continuous intravenous inotropic support may be reasonable in those hospitalized patients presenting with documented severe systolic dysfunction who present with low blood pressure and significantly depressed cardiac output to maintain systemic perfusion and preserve end-organ performance (Abraham et al., 2005; Cuffe et al., 2002; Elkayam et al., 2007). (*Level of Evidence: B*)
2. Long-term, continuous intravenous inotropic support may be considered as palliative therapy for symptom control in select patients with stage D HF despite optimal GDMT and device therapy who are not eligible for either MCS or cardiac transplantation (O'Connor et al., 1999; Hershberger et al., 2003; Gorodeski et al., 2009). (*Level of Evidence: B*)

Class III: Harm

1. Long-term use of either continuous or intermittent, intravenous parenteral positive inotropic agents, in the absence of specific indications or for reasons other than palliative care, is potentially harmful in the patient with HF ("Xamoterol in severe heart failure, 1990; Cohn et al., 1998; Hampton et al., 1997; Lubsen et al., 1996; Packer et al., 1991; Metra et al., 2009; Oliva et al., 2009). (*Level of Evidence: B*)
2. Use of parenteral inotropic agents in hospitalized patients without documented severe systolic dysfunction, low blood pressure, or impaired perfusion and evidence of significantly depressed cardiac output, with or without congestion, is potentially harmful (Abraham et al., 2005; Cuffe et al., 2002; Elkayam et al., 2007). (*Level of Evidence: B*)

Mechanical Circulatory Support

Class IIa

1. MCS is beneficial in carefully selected‡ patients with stage D HF*r*EF in whom definitive management (e.g., cardiac transplantation) or cardiac recovery is anticipated or planned (Pagani et al., 2009; Alba et al., 2010; Elhenawy et al., 2011; Nair et al., 2010; Miller et al.,2007; Lahpor et al., 2010; Starling et al., 2011; Grady et al., 2004). (*Level of Evidence: B*)
2. Nondurable MCS, including the use of percutaneous and extracorporeal ventricular assist devices (VADs), is reasonable as a "bridge to recovery" or "bridge to decision" for carefully selected‡ patients with HF*r*EF with acute, profound hemodynamic compromise (Burkhoff et al., 2006; Greenberg et al., 2008; Seyfarth et al., 2008; Thiele et al., 2005). (*Level of Evidence: B*)
3. Durable MCS is reasonable to prolong survival for carefully selected‡ patients with stage D HF*r*EF (Rose et al., 2001; Stevenson et al., 2004; Rogers et al., 2007; Slaughter et al., 2009). (*Level of Evidence: B*)

‡Although optimal patient selection for MCS remains an active area of investigation, general indications for referral for MCS therapy include patients with LVEF <25% and NYHA class III–IV functional status despite GDMT, including, when indicated, CRT, with either high predicted 1-

to 2-year mortality (e.g., as suggested by markedly reduced peak oxygen consumption and clinical prognostic scores) or dependence on continuous parenteral inotropic support. Patient selection requires a multidisciplinary team of experienced advanced HF and transplantation cardiologists, cardiothoracic surgeons, nurses, and ideally, social workers and palliative care clinicians.

Cardiac Transplantation

Class I

1. Evaluation for cardiac transplantation is indicated for carefully selected patients with stage D HF despite GDMT, device, and surgical management (Mehra et al., 2006). (*Level of Evidence: C*)

The Hospitalized Patient

Precipitating Causes of Decompensated HF

Class I

1. ACS precipitating acute HF decompensation should be promptly identified by ECG and serum biomarkers, including cardiac troponin testing, and treated optimally as appropriate to the overall condition and prognosis of the patient. (*Level of Evidence: C*)
2. Common precipitating factors for acute HF should be considered during initial evaluation, as recognition of these conditions is critical to guide appropriate therapy. (*Level of Evidence: C*)

Maintenance of GDMT during Hospitalizations

Class I

1. In patients with HF*r*EF experiencing a symptomatic exacerbation of HF requiring hospitalization during chronic maintenance treatment with GDMT, it is recommended that GDMT be continued in the absence of hemodynamic instability or contraindications (Fonarow et al. "Influence," 2008; Metra et al., 2007; Butler et al., 2006). (*Level of Evidence: B*)
2. Initiation of beta-blocker therapy is recommended after optimization of volume status and successful discontinuation of intravenous diuretics, vasodilators, and inotropic agents. Beta-blocker therapy should be initiated at a low dose and only in stable patients. Caution should be used when initiating beta blockers in patients who have required inotropes during their hospital course (Fonarow et al. "Influence," 2008; Metra et al., 2007; Butler et al., 2006). (*Level of Evidence: B*)

Diuretics in Hospitalized Patients

Class I

1. Patients with HF admitted with evidence of significant fluid overload should be promptly treated with intravenous loop diuretics to reduce morbidity (Maisel et al., 2008; Peacock et al., 2007). (*Level of Evidence: B*)
2. If patients are already receiving loop diuretic therapy, the initial intravenous dose should equal or exceed their chronic oral daily dose and should be given as either intermittent boluses or continuous infusion. Urine output and signs and symptoms of congestion should be serially assessed, and the diuretic dose should be adjusted accordingly to relieve symptoms, reduce volume excess, and avoid hypotension (Felker et al., 2011). (*Level of Evidence: B*)
3. The effect of HF treatment should be monitored with careful measurement of fluid intake and output, vital signs, body weight that is determined at the same time each day, and clinical signs and symptoms of systemic perfusion and congestion. Daily serum electrolytes, urea nitrogen, and creatinine concentrations should be measured during the use of intravenous diuretics or active titration of HF medications. (*Level of Evidence: C*)

Class IIa

1. When diuresis is inadequate to relieve symptoms, it is reasonable to intensify the diuretic regimen using either:
    a. Higher doses of intravenous loop diuretics (Hunt et al., 2009; Felker et al., 2011) (*Level of Evidence: B*); or
    b. Addition of a second (e.g., thiazide) diuretic (Grosskopf, Rabinovitz, & Rosenfeld, 1986; Channer et al., 1994; Sigurd, Olesen, & Wennevold, 1975; Rosenberg et al., 2005). (*Level of Evidence: B*).

Class IIb

1. Low-dose dopamine infusion may be considered in addition to loop diuretic therapy to improve diuresis and better preserve renal function and renal blood flow (Giamouzis et al., 2010; Elkayam et al., 2008). (*Level of Evidence: B*)

Renal Replacement Therapy—Ultrafiltration

Class IIb

1. Ultrafiltration may be considered for patients with obvious volume overload to alleviate congestive symptoms and fluid weight (Costanzo et al., 2007). (*Level of Evidence: B*)
2. Ultrafiltration may be considered for patients with refractory congestion not responding to medical therapy. (*Level of Evidence: C*)

Parenteral Therapy in Hospitalized HF

Class IIb

1. If symptomatic hypotension is absent, intravenous nitroglycerin, nitroprusside, or nesiritide may be considered an adjuvant to diuretic therapy for relief of dyspnea in patients admitted with acutely decompensated HF (Colucci et al., 2000; Cioffi et al., 2003; O'Connor et al., 2011; Publication Committee for the VMAC Investigators, 2002). (*Level of Evidence: A*)

Venous Thromboembolism Prophylaxis in Hospitalized Patients

Class I

1. A patient admitted to the hospital with decompensated HF should receive venous thromboembolism prophylaxis with an anticoagulant medication if the risk–benefit ratio is favorable (Guyatt et al., 2012; Alikhan et al., 2003). (*Level of Evidence: B*)

Arginine Vasopressin Antagonists

Class IIb

1. In patients hospitalized with volume overload, including HF, who have persistent severe hyponatremia and are at risk for or having active cognitive symptoms despite water restriction and maximization of GDMT, vasopressin antagonists may be considered in the short term to improve serum sodium concentration in hypervolemic, hyponatremic states with either a V2 receptor selective or a nonselective vasopressin antagonist (Ghali et al., 2006; Schrier et al., 2006). (*Level of Evidence: B*)

Inpatient and Transitions of Care

Class I

1. The use of performance improvement systems and/or evidence-based systems of care is recommended in the hospital and early post discharge outpatient setting to identify appropriate HF patients for GDMT, provide clinicians with useful reminders to advance GDMT, and assess the clinical response (McAlister et al., 2004; Koelling et al., 2005; Fonarow et al., "Temporal," 2007; Naylor et al., 1994; Naylor et al., 2004; Fonarow et al., "Influence," 2007; Lappe et al., 2004; Phillips et al., 2004). (*Level of Evidence: B*)
2. Throughout the hospitalization as appropriate, before hospital discharge, at the first postdischarge visit, and in subsequent follow-up visits, the following should be addressed (O'Connor et al., 2008; Lappe et al., 2004; Gislason et al., 2007; Masoudi et al., 2004; Braunstein et al., 2003). (*Level of Evidence: B*):
    a. Initiation of GDMT if not previously established and not contraindicated
    b. Precipitant causes of HF, barriers to optimal care transitions, and limitations in postdischarge support
    c. Assessment of volume status and supine/upright hypotension with adjustment of HF therapy as appropriate
    d. Titration and optimization of chronic oral HF therapy
    e. Assessment of renal function and electrolytes where appropriate
    f. Assessment and management of comorbid conditions
    g. Reinforcement of HF education, self-care, emergency plans, and need for adherence
    h. Consideration for palliative care or hospice care in selected patients

3. Multidisciplinary HF disease-management programs are recommended for patients at high risk for hospital readmission, to facilitate the implementation of GDMT, to address different barriers to behavioral change, and to reduce the risk of subsequent rehospitalization for HF (McAlister et al., 2004; Windham, Bennett, & Gottlieb, 2003; Fonarow et al., 2010; Fonarow et al., "Association," 2007). (*Level of Evidence: B*)

Class IIa

1. Scheduling an early follow-up visit (within 7 to 14 days) and early telephone follow-up (within 3 days) of hospital discharge are reasonable

(Krumholz et al., 2000; Hernandez et al., 2010). (*Level of Evidence: B*)
2. Use of clinical risk-prediction tools and/or biomarkers to identify patients at higher risk for postdischarge clinical events is reasonable (Kociol et al., 2011). (*Level of Evidence: B*)

Surgical/Percutaneous/Transcatheter Interventional Treatments of HF

Class I

1. Coronary artery revascularization via coronary artery bypass graft surgery (CABG) or percutaneous intervention is indicated for patients (HF*p*EF and HF*r*EF) on GDMT with angina and suitable coronary anatomy, especially for a left main stenosis (>50%) or left main equivalent disease (American College of Cardiology Foundation et al. "ACCF/AHA/SCAI guideline for coronary artery bypass graft surgery," 2011; American College of Cardiology Foundation et al. "2011 ACCF/AHA/SCAI guideline for percutaneous coronary intervention," 2011; Filn et al., 2012; Caracciolo et al., 1995). (*Level of Evidence: C*)

Class IIa

1. CABG to improve survival is reasonable in patients with mild to moderate LV systolic dysfunction (EF 35% to 50%) and significant (≥70% diameter stenosis) multivessel CAD or proximal left anterior descending coronary artery stenosis when viable myocardium is present in the region of intended revascularization (Caracciolo et al., 1995; VA Coronary Artery Bypass Surgery Cooperative Study Group, 1992;"Eleven-year survival," 1984). (*Level of Evidence: B*)
2. CABG or medical therapy is reasonable to improve morbidity and cardiovascular mortality for patients with severe LV dysfunction (EF <35%), HF, and significant CAD (Velazquez et al., 2011; Cleland et al., 2011). (*Level of Evidence: B*)
3. Surgical aortic valve replacement is reasonable for patients with critical aortic stenosis and a predicted surgical mortality of no greater than 10% (Smith et al., "Transcatheter," 2011). (*Level of Evidence: B*)
4. Transcatheter aortic valve replacement after careful candidate consideration is reasonable for patients with critical aortic stenosis who are deemed inoperable (Leon et al., 2010). (*Level of Evidence: B*)

Class IIb

1. CABG may be considered with the intent of improving survival in patients with ischemic heart disease with severe LV systolic dysfunction (EF <35%) and operable coronary anatomy whether or not viable myocardium is present (Alderman et al., 1983; Patel et al., 2009; Velazquez et al., 2011). (*Level of Evidence: B*)
2. Transcatheter mitral valve repair or mitral valve surgery for functional mitral insufficiency is of uncertain benefit and should only be considered after careful candidate selection and with a background of GDMT (Feldman et al., 2011; Chan et al., 2012; Fattouch et al., 2009; Franzen et al., 2011). (*Level of Evidence: B*)
3. Surgical reverse remodeling or LV aneurysmectomy may be considered in carefully selected patients with HF*r*EF for specific indications, including intractable HF and ventricular arrhythmias (Jones et al., 2009). (*Level of Evidence: B*)

Coordinating Care for Patients with Chronic HF

Class I

1. Effective systems of care coordination with special attention to care transitions should be deployed for every patient with chronic HF that facilitate and ensure effective care that is designed to achieve GDMT and prevent hospitalization (Inglis et al., 2010; McAlister et al., 2004; Naylor et al., 2004; Coleman, Boult & American Geriatrics Society Health Care Systems Committee, 2003; Stewart, Person, & Horowitz, 1998; Stewart, Marley, & Horowitz, 1999; Sochalski et al., 2009; Laramee et al., 2003; Clark et al., 2007; Chaudhry et al., 2007; Riegel et al., 2002; Riegel et al., 2006; Krumholz et al., 2006; Faxon et al., 2004; Rich et al.,1995; McAlister et al., 2001; Riegel & LePetri, 2001; Coleman, Mahoney, & Parry, 2005). (*Level of Evidence: B*)
2. Every patient with HF should have a clear, detailed, and evidence-based plan of care that ensures the achievement of GDMT goals, effective management of comorbid conditions, timely follow-up with the healthcare team, appropriate dietary and physical activities, and compliance with secondary prevention guidelines for cardiovascular disease. This plan of care should be updated regularly and made readily available to all members of each patient's healthcare team (Smith et al., "AHA/ACCF," 2011). (*Level of Evidence: C*)
3. Palliative and supportive care is effective for patients with symptomatic advanced HF to improve quality of life (Allen et al., 2012; Lorenz et al., 2008; Hauptman & Havranek, 2005; Adler et al., 2009; Qaseem et al., 2008). (*Level of Evidence: B*)

Quality Metrics/Performance Measures

Class I

1. Performance measures based on professionally developed clinical practice guidelines should be used with the goal of improving quality of care for HF (Fonarow et al., "Temporal trends," 2007; Fonarow et al., 2010; Jencks, Huff, & Cuerdon, 2003). (*Level of Evidence: B*)

Class IIa

1. Participation in quality improvement programs and patient registries based on nationally endorsed, clinical practice guideline–based quality and performance measures can be beneficial in improving the quality of HF care (Fonarow et al., "Temporal trends," 2007; Fonarow et al., 2010). (*Level of Evidence: B*)

Definitions:

Applying Classification of Recommendations and Level of Evidence

		Size of Treatment Effect					
		CLASS I *Benefit >>> Risk* Procedure/Treatment SHOULD be performed/ administered	CLASS IIa *Benefit >> Risk* *Additional studies with focused objectives needed* IT IS REASONABLE to perform procedure/administer treatment	CLASS IIb *Benefit ≥ Risk* *Additional studies with broad objectives needed; additional registry data would be helpful* Procedure/Treatment MAY BE CONSIDERED	CLASS III *No Benefit* or Class III *Harm*		
						Procedure/Test	Treatment
					COR III: No Benefit	Not helpful	No proven benefit
					COR III: Harm	Excess cost without benefit or harmful	Harmful to patients
Estimate of Certainty (Precision) of Treatment Effect	LEVEL A Multiple populations evaluated* Data derived frommultiple randomized clinical trials or meta-analyses	• Recommendation that procedure or treatment is useful/effective • Sufficient evidence frommultiple randomized trials or meta-analyses	• Recommendation in favor oftreatment or procedure being useful/effective • Some conflicting evidence frommultiple randomized trials or meta-analyses	• Recommendation's usefulness/efficacy less well established • Greater conflicting evidence frommultiple randomized trials or meta-analyses	• Recommendation that procedure or treatment is not useful/effective and may be harmful • Sufficient evidence frommultiple randomized trials or meta-analyses		
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	LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies or standard ofcare	• Recommendation that procedure or treatment is useful/effective • Only expert opinion, case studies, or standard ofcare	• Recommendation in favor oftreatment or procedure being useful/effective • Only diverging expert opinion, case studies, or standard ofcare	• Recommendation's usefulness/efficacy less well established • Only diverging expert opinion, case studies, or standard ofcare	• Recommendation that procedure or treatment is not useful/effective and may be harmful • Only expert opinion, case studies, or standard ofcare		

A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Although randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as gender, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use.

Clinical Algorithm(s)

The following algorithms are provided in the original guideline document:

- Stage C HF*r*EF: evidence-based, guideline-directed medical therapy
- Indications for CRT
- Stages in the development of HF and recommended therapy by stage
- Pharmacological management of patients with newly discovered AF
- Pharmacological management of patients with recurrent paroxysmal AF

Scope

Disease/Condition(s)

Heart failure (HF) including heart failure with reduced ejection fraction (HF*r*EF) and heart failure with preserved ejection fraction (HF*p*EF)

Other Disease/Condition(s) Addressed

Sleep disorders

Guideline Category

Diagnosis

Evaluation

Management

Prevention

Risk Assessment

Treatment

Clinical Specialty

Cardiology

Family Practice

Geriatrics

Internal Medicine

Thoracic Surgery

Intended Users

Advanced Practice Nurses

Health Care Providers

Nurses

Physician Assistants

Physicians

Guideline Objective(s)

- To assist clinicians in clinical decision making by describing a range of generally acceptable approaches to the diagnosis, management, and prevention of heart failure (HF)
- To define practices that meet the needs of most patients in most circumstances

Target Population

Adults with heart failure (HF)

Note: Although of increasing importance, HF in children and congenital heart lesions in adults are not specifically addressed in this guideline.

Interventions and Practices Considered

Diagnosis/Evaluation

1. Thorough history and physical examination, including family history, vital signs, and volume status
2. Risk assessment
3. Laboratory testing: complete blood count, urinalysis, serum electrolytes (including calcium and magnesium), blood urea nitrogen, serum creatinine, fasting blood glucose, lipid profile, liver function tests, and thyroid-stimulating hormone
4. Serial monitoring to include serum electrolytes and renal function during treatment
5. 12-lead electrocardiography
6. Screening for hemochromatosis or human immunodeficiency virus (HIV) in selected patients
7. Diagnostic tests for rheumatologic diseases, amyloidosis, or pheochromocytoma, if indicated
8. Measurement of B-type natriuretic peptide (BNP) and N-terminal pro-B-type natriuretic peptide (NT-proBNP) and/or cardiac troponin
9. Measurement of other biomarkers of myocardial injury or fibrosis
10. Chest x-ray
11. Two-dimensional echocardiogram with Doppler
12. Radionuclide ventriculography, if echocardiogram not available
13. Magnetic resonance imaging in selected patients
14. Invasive hemodynamic monitoring (catheterization) to guide therapy in selected patients
15. Coronary arteriography in selected patients
16. Endomyocardial biopsy when a specific diagnosis is suspected that would influence therapy

Treatment/Management*

1. General management
    - Recognition and treatment of elevated blood pressure
    - Management of dyslipidemia and vascular risk
    - Management of obesity and diabetes mellitus
    - Recognition and control of other conditions that may lead to heart failure (HF) (e.g., tobacco use, known cardiotoxic agents, sleep apnea)
2. Pharmacological interventions
    - Diuretics
    - Angiotensin-converting-enzyme (ACE) inhibitors
    - Angiotensin receptor blockers (ARBs)
    - Beta blockers
    - Hydralazine and isosorbide dinitrate
    - Digoxin
    - Anticoagulant therapy
    - Omega-3 polyunsaturated fatty acid (PUFA) supplementation
    - Aldosterone receptor antagonists
    - Infusion of a positive inotropic drug only as palliation for patients with end-stage disease or while waiting for heart transplantation
    - Nutritional supplements and hormonal therapy (of no benefit and not recommended)
    - Calcium channel–blocking drugs (not recommended as routine treatment for patients with heart failure with reduced ejection fraction [HF*r*EF])
3. Nonpharmacological management
    - Patient education on self-care
    - Sodium restriction
    - Treatment of sleep disorders
    - Activity, exercise prescription, and cardiac rehabilitation

- Fluid restriction (in stage D HF)

4. Device therapy
    - Implantable cardioverter-defibrillator (ICD) therapy
    - Cardiac resynchronization therapy
5. Surgical/percutaneous/transcatheter interventional treatments of HF
    - Coronary artery bypass graft (CABG) surgery or percutaneous coronary artery intervention
    - Surgical aortic valve replacement in selected patients
    - Transcatheter aortic valve replacement in selected patients
    - Transcatheter mitral valve repair in selected patients
    - Surgical reverse remodeling or left ventricular aneurysmectomy in selected patients
    - Heart transplantation
    - Mechanical circulatory support (MCS)

*See the "Major Recommendations" field for interventions for the hospitalized patient, coordination of care for chronic HF, and quality metrics/performance measures.

Major Outcomes Considered

- Sensitivity and specificity of diagnostic instruments
- Morbidity and mortality due to heart failure (HF)
- Symptoms of HF
- Cardiovascular events
- Risk of HF
- Risk of death and hospitalization
- Survival rates
- Quality of life and sense of well-being
- Adverse effects

Methodology

Methods Used to Collect/Select the Evidence

Searches of Electronic Databases

Description of Methods Used to Collect/Select the Evidence

An extensive evidence review was conducted through October 2011 and includes selected other references through April 2013. Searches were extended to studies, reviews, and other evidence conducted in human subjects and that were published in English from PubMed, EMBASE, Cochrane, Agency for Healthcare Research and Quality Reports, and other selected databases relevant to this guideline. Key search words included but were not limited to the following: *heart failure, cardiomyopathy, quality of life, mortality, hospitalizations, prevention, biomarkers, hypertension, dyslipidemia, imaging, cardiac catheterization, endomyocardial biopsy, angiotensin-converting enzyme inhibitors, angiotensin-receptor antagonists/blockers, beta blockers, cardiac, cardiac resynchronization therapy, defibrillator, device-based therapy, implantable cardioverter defibrillator, device implantation, medical therapy, acute decompensated heart failure, preserved ejection fraction, terminal care and transplantation, quality measures*, and *performance measures*. Additionally, the committee reviewed documents related to the subject matter previously published by the American College of Cardiology Foundation (ACCF) and American Heart Association (AHA). References elected and published in this document are representative and not all-inclusive.

Number of Source Documents

1365

Methods Used to Assess the Quality and Strength of the Evidence

Weighting According to a Rating Scheme (Scheme Given)

Rating Scheme for the Strength of the Evidence

Applying Classification of Recommendations and Level of Evidence

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A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Although randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as sex, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use.

Methods Used to Analyze the Evidence

Review of Published Meta-Analyses

Systematic Review with Evidence Tables

Description of the Methods Used to Analyze the Evidence

In analyzing the data and developing recommendations and supporting text, the writing committee uses evidence-based methodologies developed by the Task Force. The Level of Evidence (LOE) is an estimate of the certainty or precision of the treatment effect. The writing committee reviews and ranks evidence supporting each recommendation with the weight of evidence ranked as LOE A, B, or C according to specific definitions that are included in the "Rating Scheme for the Strength of the Evidence" field. Studies are identified as observational, retrospective, prospective, or randomized where appropriate.

To provide clinicians with a representative evidence base, whenever deemed appropriate or when published, the absolute risk difference and number needed to treat or harm are provided in the guideline (within tables), along with confidence intervals and data related to the relative treatment effects such as odds ratio, relative risk, hazard ratio, and incidence rate ratio.

Methods Used to Formulate the Recommendations

Expert Consensus

Description of Methods Used to Formulate the Recommendations

Experts in the subject under consideration are selected by the American College of Cardiology Foundation (ACCF) and American Heart Association (AHA) to examine subject-specific data and write guidelines in partnership with representatives from other medical organizations and specialty groups. Writing committees are asked to perform a literature review; weigh the strength of evidence for or against particular tests, treatments, or procedures; and include estimates of expected outcomes where such data exist. Patient-specific modifiers, comorbidities, and issues of patient preference that may influence the choice of tests or therapies are considered. When available, information from studies on cost is considered, but data on efficacy and outcomes constitute the primary basis for the recommendations contained herein.

In analyzing the data and developing recommendations and supporting text, the writing committee uses evidence-based methodologies developed by the Task Force. The Class of Recommendation (COR) is an estimate of the size of the treatment effect considering risks versus benefits in addition to evidence and/or agreement that a given treatment or procedure is or is not useful/effective or in some situations may cause harm. The writing committee reviews and ranks evidence supporting each recommendation with the weight of evidence ranked as LOE A, B, or C according to specific definitions that are included in the "Rating Scheme for the Strength of the Evidence" field. Studies are identified as observational, retrospective, prospective, or randomized where appropriate. For certain conditions for which inadequate data are available, recommendations are based on expert consensus and clinical experience and are ranked as LOE C. When recommendations at LOE C are supported by historical clinical data, appropriate references (including clinical reviews) are cited if available.

For issues for which sparse data are available, a survey of current practice among the clinicians on the writing committee is the basis for LOE C recommendations and no references are cited. The schema for COR and LOE are summarized in the "Rating Scheme for the Strength of the Evidence" field. Table 1 in the original guideline document provides suggested phrases for writing recommendations within each COR. A new addition to this methodology is separation of the Class III recommendations to delineate whether the recommendation is determined to be of "no benefit" or is associated with "harm" to the patient. In addition, in view of the increasing number of comparative effectiveness studies, comparator verbs and suggested phrases for writing recommendations for the comparative effectiveness of one treatment or strategy versus another have been added for COR I and IIa, LOE A or B only.

In view of the advances in medical therapy across the spectrum of cardiovascular diseases, the Task Force has designated the term *guideline-directed medical therapy (GDMT)* to represent optimal medical therapy as defined by ACCF/AHA guideline–recommended therapies (primarily Class I). This new term, *GDMT*, will be used herein and throughout all future guidelines.

Because the ACCF/AHA practice guidelines address patient populations (and clinicians) residing in North America, drugs that are not currently available in North America are discussed in the text without a specific COR. For studies performed in large numbers of subjects outside North America, each writing committee reviews the potential influence of different practice patterns and patient populations on the treatment effect and relevance to the ACCF/AHA target population to determine whether the findings should inform a specific recommendation.

Organization of the Writing Committee

The committee was composed of physicians and a nurse with broad expertise in the evaluation, care, and management of patients with heart failure (HF). The authors included general cardiologists, HF and transplant specialists, electrophysiologists, general internists, and physicians with methodological expertise. The committee included representatives from the ACCF, AHA, American Academy of Family Physicians, American College of Chest Physicians, American College of Physicians, Heart Rhythm Society, and International Society for Heart and Lung Transplantation.

Scope of This Guideline with Reference to Other Relevant Guidelines or Statements

This guideline covers multiple management issues for the adult patient with HF. Although there is an abundance of evidence addressing HF, for many important clinical considerations, this writing committee was unable to identify sufficient data to properly inform a recommendation. The writing committee actively worked to reduce the number of LOE "C" recommendations, especially for Class I-recommended therapies. Despite

these limitations, it is apparent that much can be done for HF. Adherence to the clinical practice guidelines herein reproduced should lead to improved patient outcomes.

Although of increasing importance, HF in children and congenital heart lesions in adults are not specifically addressed in this guideline. The reader is referred to publicly available resources to address questions in these areas. However, this guideline does address HF with preserved ejection fraction (EF) in more detail and similarly revisits patients hospitalized with HF. Additional areas of renewed interest are in stage D HF, palliative care, transition of care, and quality of care for HF. Certain management strategies appropriate for the patient at risk for HF or already affected by HF are also reviewed in numerous relevant clinical practice guidelines and scientific statements published by the ACCF/AHA Task Force on Practice Guidelines, AHA, ACCF Task Force on Appropriate Use Criteria, European Society of Cardiology, Heart Failure Society of America, and the National Heart, Lung, and Blood Institute. The writing committee saw no need to reiterate the recommendations contained in those guidelines and chose to harmonize recommendations when appropriate and eliminate discrepancies. This is especially the case for device-based therapeutics, where complete alignment between the HF guideline and the device-based therapy guideline was deemed imperative. Some recommendations from earlier guidelines have been updated as warranted by new evidence or a better understanding of earlier evidence, whereas others that were no longer accurate or relevant or which were overlapping were modified; recommendations from previous guidelines that were similar or redundant were eliminated or consolidated when possible.

The present document recommends a combination of lifestyle modifications and medications that constitute GDMT. GDMT is specifically referenced in the recommendations for the treatment of HF (Section 7.3.2 in the original guideline document). Both for GDMT and other recommended drug treatment regimens, the reader is advised to confirm dosages with product insert material and to evaluate carefully for contraindications and drug-drug interactions. Table 2 in the original guideline document is a list of documents deemed pertinent to this effort and is intended for use as a resource; it obviates the need to repeat already extant guideline recommendations. Additional other HF guideline statements are highlighted as well for the purpose of comparison and completeness.

Rating Scheme for the Strength of the Recommendations

See "Rating Scheme for the Strength of the Evidence" field, above.

Cost Analysis

The guideline developers reviewed published cost analyses.

Method of Guideline Validation

External Peer Review

Internal Peer Review

Description of Method of Guideline Validation

This document was reviewed by 2 official reviewers each nominated by both the American College of Cardiology Foundation (ACCF) and the American Heart Association (AHA), as well as 1 to 2 reviewers each from the American Academy of Family Physicians, American College of Chest Physicians, Heart Rhythm Society, and International Society for Heart and Lung Transplantation, as well as 32 individual content reviewers (including members of the ACCF Adult Congenital and Pediatric Cardiology Council, ACCF Cardiovascular Team Council, ACCF Council on Cardiovascular Care for Older Adults, ACCF Electrophysiology Committee, ACCF Heart Failure and Transplant Council, ACCF Imaging Council, ACCF Prevention Committee, ACCF Surgeons' Scientific Council, and ACCF Task Force on Appropriate Use Criteria).

This document was approved for publication by the governing bodies of the ACCF and AHA and endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation, American College of Chest Physicians, Heart Rhythm Society, and International Society for Heart and Lung Transplantation.

This document was approved by the American College of Cardiology Foundation Board of Trustees and the American Heart Association Science Advisory and Coordinating Committee in May 2013.

Evidence Supporting the Recommendations

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Type of Evidence Supporting the Recommendations

The type of supporting evidence is identified and graded for each recommendation (see the "Major Recommendations" field).

Benefits/Harms of Implementing the Guideline Recommendations

Potential Benefits

Appropriate and effective diagnosis and management of patients with heart failure (HF)

Potential Harms

- Routine endomyocardial biopsy is not recommended in all cases of heart failure (HF), given limited diagnostic yield and the risk of procedure-related complications.
- Regardless of their mechanism of action (e.g., inhibition of phosphodiesterase, stimulation of adrenergic or dopaminergic receptors, calcium sensitization), chronic oral inotrope treatment increased mortality, mostly related to arrhythmic events. Inotropes should be considered only in such patients with systolic dysfunction who have low cardiac index and evidence of systemic hypoperfusion and/or congestion. To minimize adverse effects, lower doses are preferred. Similarly, the ongoing need for inotropic support and the possibility of discontinuation should be regularly assessed. See Table 26 in the original guideline document for adverse effects of intravenous inotropic agents.
- The principal adverse effects of diuretics include electrolyte and fluid depletion, as well as hypotension and azotemia. Diuretics can cause the depletion of potassium and magnesium, which can predispose patients to serious cardiac arrhythmias. The risk of electrolyte depletion is markedly enhanced when 2 diuretics are used in combination.
- The majority of the adverse reactions of angiotensin-converting enzyme (ACE) inhibitors can be attributed to the 2 principal pharmacological actions of these drugs: those related to angiotensin suppression and those related to kinin potentiation. Other types of adverse effects may also occur (e.g., rash and taste disturbances). Up to 20% of patients will experience an ACE inhibitor–induced cough. With the use of ACE inhibitors, particular care should be given to the patient's volume status, renal function, and concomitant medications. However, most HF patients (85% to 90%) can tolerate these drugs. Clinicians should prescribe an ACE inhibitor with caution if the patient has very low systemic blood pressures (systolic blood pressure <80 mm Hg), markedly increased serum levels of creatinine (>3 mg/dL), bilateral renal artery stenosis, or elevated levels of serum potassium (>5.0 mEq/L).
- Initiation of treatment with a beta blocker may produce 4 types of adverse reactions that require attention and management: fluid retention and worsening HF; fatigue; bradycardia or heart block; and hypotension. The occurrence of fluid retention or worsening HF is not generally a reason for the permanent withdrawal of treatment. Such patients generally respond favorably to intensification of conventional therapy, and once treated, they remain excellent candidates for long-term treatment with a beta blocker. The slowing of heart rate and cardiac conduction produced by beta blockers is generally asymptomatic and thus requires no treatment; however, if the bradycardia is accompanied by dizziness or lightheadedness or if second- or third-degree heart block occurs, clinicians should decrease the dose of the beta blocker. Clinicians may minimize the risk of hypotension by administering the beta blocker and ACE inhibitor at different times during the day. Hypotensive symptoms may also resolve after a decrease in the dose of diuretics in patients who are volume depleted. If hypotension is accompanied by other clinical evidence of hypoperfusion, beta-blocker therapy should be decreased or discontinued pending further patient evaluation. The symptom of fatigue is multifactorial and is perhaps the hardest symptom to address with confidence. Although fatigue may be related to beta blockers, other causes of fatigue should be considered, including sleep apnea, overdiuresis, or depression.
- Adherence to the combination of hydralazine and isosorbide dinitrate has generally been poor because of the large number of tablets required, frequency of administration, and the high incidence of adverse reactions. Frequent adverse effects include headache, dizziness, and gastrointestinal complaints. Nevertheless, the benefit of these drugs can be substantial and warrant a slower titration of the drugs to enhance tolerance of the therapy.
- The principal adverse reactions to digoxin occur primarily when it is administered in large doses, especially in the elderly, but large doses are not necessary for clinical benefits. The major adverse effects include cardiac arrhythmias (e.g., ectopic and re-entrant cardiac rhythms and heart block), gastrointestinal symptoms (e.g., anorexia, nausea, and vomiting), and neurological complaints (e.g., visual disturbances, disorientation, and confusion). Overt digoxin toxicity is commonly associated with serum digoxin levels >2 ng/mL. However, toxicity may also occur with lower digoxin levels, especially if hypokalemia, hypomagnesemia, or hypothyroidism coexists. The concomitant use of clarithromycin, dronedarone, erythromycin, amiodarone, itraconazole, cyclosporine, propafenone, verapamil, or quinidine can increase serum digoxin concentrations and may increase the likelihood of digoxin toxicity. The dose of digoxin should be reduced if treatment with these drugs is initiated. In addition, a low lean body mass and impaired renal function can also elevate serum digoxin levels, which may explain the increased risk of digoxin toxicity in elderly patients.
- Trials of newer oral anticoagulants have compared efficacy and safety with warfarin therapy rather than placebo. Several new oral anticoagulants are now available, including the factor Xa inhibitors apixaban and rivaroxaban and the direct thrombin inhibitor dabigatran. These drugs have few food and drug interactions compared with warfarin and no need for routine coagulation monitoring or dose adjustment. The fixed dosing together with fewer interactions may simplify patient management, particularly with the polypharmacy commonly seen in HF. These drugs have a potential for an improved benefit–risk profile compared with warfarin, which may increase their use in practice, especially in those at increased bleeding risk. However, important adverse effects have been noted with these new anticoagulants, including gastrointestinal distress, which may limit compliance. At present, there is no commercially available agent to reverse the effect of these newer drugs. Trials comparing new anticoagulants with warfarin have enrolled >10,000 patients with HF. As more detailed evaluations of the comparative benefits and risks of these newer agents in patients with HF are still pending, the writing committee considered their use in patients with HF and nonvalvular AF as an alternative to warfarin to be reasonable.
- Because of possible adverse effects and drug interactions of nutritional supplements and their widespread use, clinicians caring for patients with HF should routinely inquire about their use. Until more data are available, nutritional supplements or hormonal therapies are not recommended for the treatment of HF.

- Because nesiritide has a longer effective half-life than nitroglycerin or nitroprusside, adverse effects such as hypotension may persist longer.

Contraindications

Contraindications

- Patients should not be given an angiotensin-converting enzyme (ACE) inhibitor if they have experienced life-threatening adverse reactions (i.e., angioedema) during previous medication exposure or if they are pregnant or plan to become pregnant.
- Because of reports of development of cardiomyopathy, sibutramine is contraindicated in heart failure.
- Sulfite allergy is a contraindication to dobutamine therapy.
- Treatment with thiazolidinediones (e.g., rosiglitazone) is associated with fluid retention in patients with heart failure and should be avoided in patients with New York Heart Association (NYHA) class II through IV heart failure.
- In patients with previously established structural heart disease, the administration of agents known to have negative inotropic properties such as nondihydropyridine calcium channel blockers and certain antiarrhythmics should be avoided.

Qualifying Statements

Qualifying Statements

- Because the American College of Cardiology Foundation (ACCF)/American Heart Association (AHA) guidelines address patient populations (and clinicians) residing in North America, drugs that are not currently available in North America are discussed in the text without a specific class of recommendation (COR). For studies performed in large numbers of subjects outside North America, each writing committee reviews the potential influence of different practice patterns and patient populations on the treatment effect and relevance to the ACCF/AHA target population to determine whether the findings should inform a specific recommendation.
- The ACCF/AHA practice guidelines are intended to assist clinicians in clinical decision making by describing a range of generally acceptable approaches to the diagnosis, management, and prevention of specific diseases or conditions. The guidelines attempt to define practices that meet the needs of most patients in most circumstances. The ultimate judgment regarding care of a particular patient must be made by the clinician and patient in light of all the circumstances presented by that patient. As a result, situations may arise for which deviations from these guidelines may be appropriate. Clinical decision making should involve consideration of the quality and availability of expertise in the area where care is provided. When these guidelines are used as the basis for regulatory or payer decisions, the goal should be improvement in quality of care. The Task Force recognizes that situations arise in which additional data are needed to inform patient care more effectively; these areas will be identified within each respective guideline when appropriate.
- Prescribed courses of treatment in accordance with these recommendations are effective only if followed. Because lack of patient understanding and adherence may adversely affect outcomes, clinicians should make every effort to engage the patient's active participation in prescribed medical regimens and lifestyles. In addition, patients should be informed of the risks, benefits, and alternatives to a particular treatment and be involved in shared decision making whenever feasible, particularly for COR IIa and IIb, for which the benefit-to-risk ratio may be lower.

Implementation of the Guideline

Description of Implementation Strategy

An implementation strategy was not provided.

Implementation Tools

Clinical Algorithm

Quick Reference Guides/Physician Guides

Slide Presentation

Tool Kits

For information about availability, see the *Availability of Companion Documents* and *Patient Resources* fields below.

Institute of Medicine (IOM) National Healthcare Quality Report Categories

IOM Care Need

End of Life Care

Living with Illness

Staying Healthy

IOM Domain

Effectiveness

Patient-centeredness

Identifying Information and Availability

Bibliographic Source(s)

Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Drazner MH, Fonarow GC, Geraci SA, Horwich T, Januzzi JL, Johnson MR, Kasper EK, Levy WC, Masoudi FA, McBride PE, McMurray JJ, Mitchell JE, Peterson PN, Riegel B, Sam F, Stevenson LW, Tang WH, Tsai EJ, Wilkoff BL. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2013 Oct 15;62(16):e147-239. [924 references] PubMed

Adaptation

Not applicable: The guideline was not adapted from another source.

Date Released

1995 Nov 1 (revised 2013 Oct 15)

Guideline Developer(s)

American College of Cardiology Foundation - Medical Specialty Society

American Heart Association - Professional Association

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Guideline Committee

2013 ACCF/AHA Guideline for the Management of Heart Failure Writing Committee

American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines

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*Former Task Force member during this writing effort.

Financial Disclosures/Conflicts of Interest

The Task Force makes every effort to avoid actual, potential, or perceived conflicts of interest that may arise as a result of industry relationships or personal interests among the members of the writing committee. All writing committee members and peer reviewers of the guideline are required to disclose all current healthcare-related relationships, including those existing 12 months before initiation of the writing effort. In December 2009, the American College of Cardiology Foundation (ACCF) and American Heart Association (AHA) implemented a new policy for relationship with industry and other entities (RWI) that requires the writing committee chair plus a minimum of 50% of the writing committee to have no *relevant* RWI (Appendix 1 in the original guideline document includes the ACCF/AHA definition of relevance). These statements are reviewed by the Task Force and all members during each conference call and/or meeting of the writing committee and are updated as changes occur. All guideline recommendations require a confidential vote by the writing committee and must be approved by a consensus of the voting members. Members are not permitted to draft or vote on any text or recommendations pertaining to their RWI.

Members who recused themselves from voting are indicated in the list of writing committee members, and specific section recusals are noted in Appendix 1 of the original guideline document. Authors' and peer reviewers' RWI pertinent to this guideline are disclosed in Appendixes 1 and 2 of the original guideline document, respectively. Additionally, to ensure complete transparency, writing committee members' comprehensive disclosure information—including RWI not pertinent to this document—is available as an online supplement. Comprehensive disclosure information for the Task Force is also available online from the American College of Cardiology Web site . The work of writing committees is supported exclusively by the ACCF and AHA without commercial support. Writing committee members volunteered their time for this activity.

Guideline Endorser(s)

American Association of Cardiovascular and Pulmonary Rehabilitation - Medical Specialty Society

American College of Chest Physicians - Medical Specialty Society

Heart Rhythm Society - Professional Association

International Society for Heart and Lung Transplantation - Professional Association

Guideline Status

This is the current release of the guideline.

This guideline updates a previous version: Hunt SA, Abraham WT, Chin MH, Feldman AM, Francis GS, Ganiats TG, Jessup M, Konstam MA, Mancini DM, Michl K, Oates JA, Rahko PS, Silver MA, Stevenson LW, Yancy CW, American College of Cardiology Foundation, American Heart Association. 2009 focused update incorporated into the ACC/AHA 2005 guidelines for the diagnosis and management of heart failure in adults [trunc]. J Am Coll Cardiol. 2009 Apr 14;53(15):e1-e90.

Guideline Availability

Electronic copies: Available in Portable Document Format (PDF) from the Journal of the American College of Cardiology (ACC) Web site and from the Circulation Web site.

Print copies: Available from the American College of Cardiology, 2400 N Street NW, Washington DC, 20037; (800) 253-4636 (US only).

Availability of Companion Documents

The following are available:

- Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Drazner MH, Fonarow GC, Geraci SA, Horwich T, Januzzi JL, Johnson MR, Kasper EK, Levy WC, Masoudi FA, McBride PE, McMurray JJV, Mitchell JE, Peterson PN, Riegel B, Sam F, Stevenson LW, Tang WHW, Tsai EJ, Wilkoff BL. 2013 ACCF/AHA guideline for the management of heart failure: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013;62:1495–539. Electronic copies: Available in Portable Document Format (PDF) from the Journal of the American College of Cardiology (JACC) Web site.
- Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Drazner MH, Fonarow GC, Geraci SA, Horwich T, Januzzi JL, Johnson MR, Kasper EK, Levy WC, Masoudi FA, McBride PE, McMurray JJV, Mitchell JE, Peterson PN, Riegel B, Sam F, Stevenson LW, Tang WHW, Tsai EJ, Wilkoff BL. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Online data supplement. J Am Coll Cardiol 2013;62:e147–239. Available in PDF from the JACC Web site.
- Methodology manual and policies from the ACCF/AHA Task Force on Practice Guidelines. 2010 Jun. 88 p. American College of Cardiology Foundation and American Heart Association, Inc. Electronic copies: Available in PDF from the American College of Cardiology (ACC) Web site.

Print copies: Available from the American College of Cardiology, 2400 N Street NW, Washington DC, 20037; (800) 253-4636 (US only).

A clinical toolkit is available from the ACC Web site. In addition, a slide set is available to subscribers from the ACC Web site.

Patient Resources

None available

NGC Status

This summary was completed by ECRI on June 30, 1998. The information was verified by the guideline developer on December 1, 1998. This NGC summary was updated on February 25, 2002. The updated information was verified by the guideline developer as of April 17, 2002. This NGC summary was updated again on September 12, 2005. The updated information was verified by the guideline developer on November 17, 2005. This summary was updated by ECRI Institute on September 5, 2007 following the U.S. Food and Drug Administration advisory on the Thiazolidinedione class of antidiabetic drugs. This summary was updated by ECRI Institute on November 28, 2007 following the U.S. Food and Drug Administration advisory on the Avandia (rosiglitazone maleate) Tablets. This summary was updated by ECRI Institute on March 10, 2008 following the U.S. Food and Drug Administration advisory on Avandia (rosiglitazone maleate). This summary was updated by ECRI Institute on November 8, 2010 following the U.S. Food and Drug Administration advisory on Avandia (rosiglitazone). This NGC summary was updated on November 18, 2010. The updated information was verified by the guideline developer on February 10, 2011. This summary was updated by ECRI Institute on July 22, 2013 following the U.S. Food and Drug administration advisory on Olmesartan Medoxomil. This summary was updated by ECRI Institute on December 6, 2013. The updated information was verified by the guideline developer on January 22, 2014.

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